HBeAg were co-incubated for 6h, compared to that of before stimulation, expression of B7-H1 on $CD14^+$ cells increased, expression of TLR2 decreased, expression of B7-H1 and PD-1 on $CD4^+$ and $CD8^+$ T cells increased significantly, expression of costimulatory molecules CD28 on $CD4^+$ cells decreased;Inflammatory factor TNF-α and anti-viral cytokine IFN-γ of HBeAg-stimulated PBMC from HBeAg-negative CHB patients, increased and reduced respectively.
Conclusion: By up-regulating expression of B7-H1 and PD-1,HBeAg may inhibit expression of TLR2 on $CD14^+$ cells, reduce expression of costimulatory molecules CD28 on T lymphocyte, inhibit polarization of Th cells, and reduce active secretion of anti-viral factor of patients, which leaded to function of specific cell-mediated immunity became low, thereby clearance of virus by T lymphocyte were suppressed, which eventually resulted in persistence of HBV infection.

PP-108 Detecting hepatitis B virus large surface protein in patients with chronic hepatitis B: a clinical study

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Objective: To investigate the clinical significance of hepatitis B virus large protein (HBLP) detection in prediction of HBV DNA replication and the effect of anti-viral treatment.
Methods: The Serum samples were collected from 90 patients with HBV infection before and after anti-viral treatment. HBV DNA level was quantitatively detected using real-time polymerase chain reaction. HBLP was measured by enzymelinked immunosorbent assay (ELISA). The collected data were analyzed by bivariate correlations method.
Results: There was no significant difference between the detectable rates of HBV DNA and HBV-LP in 90 blood samples before treatment ($p>0.05$); The levels of serum HBV-LP was positively correlated with HBV DNA copies during anti-viral treatment ($r=0.857$, $P=0.000$). The OD value of HBV-LP and the copies of HBV-DNA decreased in the same trend along with treatment.
Conclusion: HBLP expression can reflect the state of HBV DNA replication. The decrease of HBLP and HBV-DNA during the anti-viral treatment could estimate the HBV replication state and predict the effect of anti-viral treatment.

PP-109 A study for HBsAg routine test negative results

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Objective: To see the false HBsAg negative results for serum samples in daily work and to improve the clinical laboratory tests quality.
Methods: Four hundreds HBsAg negative stochastic serum samples were collected from the copy tubes in daily work for detecting hepatitis B Virus markers (HBVM) with national ELISA reagent kits divided into 200 samples with HBsAb negative and 200 positive and put them −20° frostily. HBsAg markers were counterchecked with other two adding kinds of national reagent and American MONOLISA HBsAg ULTRA reagents (total 4 kinds) at the 400 samples and filtrated the positive samples. At the last filtrated samples, HBV DNA levels were doubly quantitative analyzed with fluorescence quantitative PCR (FQ-PCR) and taking the mean results.
Results: We educed the conform HBsAg negative results from the three kinds of national reagents but five positive results from the American reagents in repeating HBsAg detection at the 200 HBsAb negative samples. No positive results were checked out from the 200 HBsAb positive samples with national or foreign reagents. The HBV DNA FQ-PCR quantitative results were all positive but less than 500 copies/ml.
Conclusion: The sensitive level of the HBsAg routine test ELISA reagents is generally on the low side and easily bring on the false HBsAg negative results and the false results are more frequently from the HBsAb negative people. This maybe connected with occult HBV infection, we should attach importance to the HBVM counterchecking work at these people.

PP-110 Detecting hepatitis B virus large surface protein to filtrate the occult HBV infection

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Objective: Detecting hepatitis B virus large surface protein (HBLP) with serological method to filtrate the occult HBV infection and study the clinical detection strategy.
Methods: Two thousands HBsAg negative stochastic serum samples were collected from the copy tubes in daily work to detect hepatitis B Virus markers (HBVM) with national ELISA reagent kits and put them −20° frostily. The 2000 samples were detected with HBLP and filtrated the positive samples. HBsAg markers were doubly counterchecked with other two adding kinds of national ELISA reagent kits (total 3 kinds) at the filtrated samples. The last samples were doubly tested again with American MONOLISA HBsAg ULTRA reagents. HBV DNA levels were quantitative analyzed with fluorescence quantitative PCR (FQ-PCR) and taking the mean results.
Results: Fifteen HBLP positive samples were detected out from the 2000 serum samples. The conform negative results were educed from the three kinds of national reagents but conform positive results from the American reagents in repeating HBsAg detection at the 15 samples. The HBV DNA FQ-PCR quantitative results were all positive but less than 500 copies/ml.
Conclusion: The false HBsAg negative results for serum samples are more generally from national reagents than from importations and HBLP results may be positive in these samples. Detecting HBLP marker is propitious to filtrate the occult HBV infection. This study provided a kind of serological reference for actively searching for the detecting strategy in occult HBV infection field.

PP-111 Evolution of hepatitis B virus in a chronic HBV-infected patient over 2 years

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Background: Evaluation of a 3-year evolution of hepatitis B virus in a chronically infected patient was conducted.
Methods: Clinic data and HBV DNA load(s) in serum were detected at three time points (1 day, 6 month and 31 month), HBV isolates were obtained at each time.The full-length HBV genome was cloned and 26 clones including three time points were randomly selected, sequenced and phylogenetically analyzed.
Results: All of the 26 clones belonged to subgenotype C2. 13 nucleotides in this subset of clones were found to be different from the published sequences of genotype C of HBV. During the 3-year evolution, nucleotides T361A, C930A, C2351T/A2353T, C2444T were the mutations been kept in and from minor to major while C339T and T770C were the new point mutations at the 3rd time point (31 month). Short fragment deletion (nt2849 to 2867) in the preS gene became dominant at the 31-month time point and had a trend to lead to large fragment deletion, which may produce presumptive P/S fusion proteins or truncated preS proteins. At the third time point, the patient's serum ALT, HBeAg and load of HBV DNA varied greatly.
Conclusion: The evolutionary data of HBV may provide clues for the interpretation of the course of HBV chronic infection and progressive pathology of liver diseases.
Acknowledgements: This study was supported by the Natural Science Foundation of Yunnan Province (granted No. 200300172),